What Is Claimed Is:

1. A method for the identification of an agent to be used in the treatment of AD and/or symptoms thereof, wherein the agent inhibits oxygen-dependent hydrogen peroxide formation activity, but does not inhibit the superoxide-dependent hydrogen peroxide formation, the method comprising:

(a) adding said agent to an Aβ-containing sample;

(b) determining whether the agent is capable of inhibiting dissolved oxygen-dependent hydrogen peroxide formation; and

(c) determining whether the agent is capable of not inhibiting the superoxide-dependent hydrogen peroxide formation.

2. The method of claim 1, wherein the method of determining whether the agent is capable of not inhibiting the superoxide-dependent hydrogen peroxide formation is conducted using pulse radiolysis or the NBT assay.

3. The method of claim 1, wherein the determination of the ability of said agent to inhibit the superoxide-dependent hydrogen peroxide formation is made by determining whether Aβ is capable of catalytically producing Cu(I), Fe(II) or H_2O_2.

4. A method for the identification of an agent to be used in the treatment of AD and/or symptoms thereof, wherein said agent is capable of altering the production of Cu(I) by Aβ, said method comprising:

(a) adding Cu(II) to a first Aβ sample;

(b) allowing said first sample to incubate for an amount of time sufficient to generate Cu(I);

(c) adding Cu(II) to a second Aβ sample, said second sample additionally comprising a candidate pharmacological agent;

(d) allowing said second sample to incubate for the same amount of time as said first sample;

(e) determining the amount of Cu(I) produced by said first sample and said second sample; and

(f) comparing the amount of Cu(I) produced by said first sample to the amount of Cu(I) produced by said second sample;

whereby a difference in the amount of Cu(I) produced by said first sample as compared to said second sample indicates that said candidate pharmacological agent has altered the production of Cu(I) by Aβ.

5. The method of claim 4, wherein the amount of Cu(I) present in said first and said second sample is determined by

(a) adding a complexing agent to said first and said second sample, wherein said complexing agent is capable of combining with Cu(I) to form a complex compound, wherein said complex compound has an optimal visible absorption wavelength;

(b) measuring the absorbencies of said first and second samples; and

(c) calculating the concentration of Cu(I) in said first and second samples using the absorbencies obtained in (b).

6. The method of claim 5, wherein said complexing agent is bathocuproinedisulfonic anion.

7. The method of claim 4, wherein said method is performed in a microtiter plate, and the absorbency measurements are performed by a plate reader.

8. The method of claim 4, wherein two or more different test candidate agents are simultaneously evaluated for an ability to alter the production of Cu(I) by Aβ.

9. The method of claim 4, wherein said first and second Aβ samples are biological samples.

10. The method of claim 9, wherein said biological samples are CSF.

11. A method for the identification of an agent to be used in the treatment of AD and/or symptoms thereof, wherein said agent is capable of altering the production of Fe(II) by Aβ, said method comprising:

(a) adding Fe(III) to a first Aβ sample;

(b) allowing said first sample to incubate for an amount of time sufficient to generate Fe(II);

(c) adding Fe(III) to a second Aβ sample, said second sample additionally comprising a candidate pharmacological agent;

(d) allowing said second sample to incubate for the same amount of time as said first sample;

(e) determining the amount of Fe(II) produced by said first sample and second sample; and

(f) comparing the amount of Fe(II) present in said first sample to the amount of Fe(II) present in said second sample;

whereby a difference in the amount of Fe(II) present in said first sample as compared to said second sample indicates that said candidate pharmacological agent has altered the production of Fe(II) by Aβ.

12. The method of claim 11, wherein the amount of Fe(II) present in said first and second samples is determined by

(a) adding a complexing agent to said first and second samples, wherein said complexing agent is capable of combining with Fe(II) to form a complex compound, wherein said complex compound has an optimal visible absorption wavelength;

(b) measuring the absorbencies of said first and second samples; and

(c) calculating the concentration of Fe(II) in said first and second samples using the absorbencies obtained in (b).

13. The method of claim 12, wherein said complexing agent is bathophenanthrolinedisulfonic (BP) anion.

14. The method of claim 11, wherein said method is performed in a microtiter plate, and the absorbency measurements are performed by a plate reader.

15. The method of claim 11, wherein two or more different test candidate agents are simultaneously evaluated for an ability to alter the production of Fe(II) by Aβ.

16. The method of claim 11, wherein said first and second Aβ samples are biological samples.

17. The method of claim 16, wherein said biological samples are CSF.

18. A method for the identification of an agent to be used in the treatment of AD and/or symptoms thereof, wherein said agent is capable of altering the production of H_2O_2 by Aβ, said method comprising:

(a) adding Cu(II) or Fe(III) to a first Aβ sample;

(b) allowing said first sample to incubate for an amount of time sufficient to generate H_2O_2;

(c) adding Cu(II) or Fe(III) to a second Aβ sample, said second sample additionally comprising a candidate pharmacological agent;

(d) allowing said second sample to incubate for the same amount of time as said first sample;

(e) determining the amount of H_2O_2 produced by said first sample and second sample; and

(f) comparing the amount of H_2O_2 present in said first sample to the amount of H_2O_2 present in said second sample;

whereby a difference in the amount of H_2O_2 present in said first sample as compared to said second sample indicates that said candidate pharmacological agent has altered the production of H_2O_2 by Aβ.

19. The method of claim 18, wherein the Aβ samples of (a) and (b) are a biological fluid.

20. The method of claim 19, wherein said biological fluid is CSF.

21. The method of claim 18, wherein the determination of the amount of H_2O_2 present in said first and second samples is determined by

(a) adding catalase to a first aliquot of said first sample in an amount sufficient to break down all of the H_2O_2 generated by said sample;

(b) adding TCEP, in an amount sufficient to capture all of the H_2O_2 generated by said samples, to

(i) a first aliquot of said first sample;

(ii) a second aliquot of said first sample; and

(iii) said second sample;

(c) incubating the samples obtained in (b) for an amount of time sufficient to allow the TCEP to capture all of the H_2O_2;

(d) adding DTNB to said samples obtained in (c);

(e) incubating said samples obtained in (d) for an amount of time sufficient to generate TMB;

(f) measuring the absorbencies at 412 nm of said samples obtained in (e); and

(g) calculating the concentration of H_2O_2 in said first and second samples using the absorbencies obtained in (f).

22. The method of claim 18, wherein said method is performed in a microtiter plate, and the absorbency measurements are performed by a plate reader.

23. The method of claim 18, wherein two or more different test candidate agents are simultaneously evaluated for an ability to alter the production of H_2O_2 by Aβ.

24. A method for the identification of an agent to be used in the treatment of AD and/or symptoms thereof, wherein said agent is capable of reducing the toxicity of Aβ, said method comprising:

(a) adding Aβ to a first cell culture;

(b) adding Aβ to a second cell culture, said second cell culture additionally containing a candidate pharmacological agent;

(c) determining the level of neurotoxicity of Aβ in said first and second samples; and

(d) comparing the level of neurotoxicity of Aβ in said first and second samples,

whereby a lower neurotoxicity level in said second sample as compared to said first sample indicates that said candidate pharmacological agent has reduced the neurotoxicity of Aβ, and is thereby capable of being used to treat AD and/or symptoms thereof.

25. The method of claim 24, wherein the neurotoxicity of Aβ is determined by using an MTT assay.

26. The method of claim 24, wherein the neurotoxicity of Aβ is determined by using an LDH release assay.

27. The method of claim 24, wherein the neurotoxicity of Aβ is determined by using a Live/Dead assay.

28. The method of claim 24, wherein the cells are rat cancer cells.

29. The method of claim 24, wherein the cells are rat primary frontal neuronal cells.

30. A kit for determining whether an agent is capable of altering the production of Cu(I) by Aβ which comprises a carrier means being compartmentalized to receive in close confinement therein one or more container means wherein

(a) the first container means contains a peptide comprising Aβ peptide;

(b) a second container means contains a Cu(II) salt; and

(c) a third container means contains BC anion.

31. The kit of claim 30, wherein said Aβ peptide is present as a solution in an aqueous buffer or a physiological solution, at a concentration from about 10 μM to about 25 μM.

32. A kit for determining whether an agent is capable of altering the production of Fe(II) by Aβ which comprises a carrier means being compartmentalized to receive in close confinement therein one or more container means wherein

(a) the first container means contains a peptide comprising Aβ peptide;

(b) a second container means contains an Fe(III) salt; and

(c) a third container means contains BP anion.

33. The kit of claim 32, wherein said Aβ peptide is present as a solution in an aqueous buffer or a physiological solution, at a concentration from about 10 μM to about 25 μM.

34. A kit for determining whether an agent is capable of altering the production of H_2O_2 by Aβ which comprises a carrier means being compartmentalized to receive in close confinement therein one or more container means wherein

(a) the first container means contains a peptide comprising Aβ peptide;

(b) a second container means contains a Cu(II) salt;

(c) a third container means contains TCEP; and

(d) a fourth container means contains DTNB.

35. The kit of claim 34, wherein said Aβ peptide is present as a solution in an aqueous buffer or a physiological solution, at a concentration from about 10 μM to about 25 μM.

36. A method for the identification of an agent to be used in the treatment of AD and/or symptoms thereof, wherein said agent is capable of inhibiting redox-reactive metal-mediated crosslinking Aβ, said method comprising:

(a) adding a redox-reactive metal to a first Aβ sample;

(b) allowing said first sample to incubate for an amount of time sufficient to allow Aβ crosslinking;

(c) adding said redox-reactive metal to a second Aβ sample, said second sample additionally comprising a candidate pharmacological agent;

(d) allowing said second sample to incubate for the same amount of time as said first sample;

(e) removing an aliquot from each of said first and second samples; and

(f) determining presence or absence of crosslinking in said first and second samples,

whereby an absence of Aβ crosslinking in said second sample as compared to said first sample indicates that said candidate pharmacological agent has inhibited Aβ crosslinking.

37. The method of claim 36, wherein at (f), a western blot analysis is performed to determine the presence or absence of crosslinking in the first and second samples.

38. A method of treating AD and/or symptoms thereof, comprising administering to a patient in need thereof an effective amount of an agent identified by the screening assay of claim 1, 4, 11, 18, 24 or 36.